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ABSTRACT

Cocaine was first used by Europeans in the nineteenth century when extract from the coca leaf was combined with various beverages. Cocaine comes as a white crystalline powder. However, a product called crack cocaine may come as an opaque crystal similar in size and shape to rock salt. A third form of cocaine is known as coca paste, which is an intermediate step in the production of cocaine. Cocaine is highly water and fat soluble and, therefore, it is readily absorbed and distributed throughout the body. Route of administration seems to be more important in determining cocaine's absorption rather than dose. It is believed that cocaine produces such a powerful and rapid dependency because it directly stimulates the pleasure centers of the brain responsible for the reinforcing properties of food, water and sex. Intranasal users do not seem to be as prone to addiction in the classic sense of the term as are those who smoke cocaine. However, they may develop a very rapid and profound psychological dependence on the drug which is just as intense as addiction to opiates or alcohol. Any user, regardless of the intensity of the use or the route of administration, is at risk of developing a problem with cocaine which may require treatment. Evaluation of cocaine abuse treatment has been sparse and no consensus exists regarding optimal treatment strategies. (LLL)

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Cocaine

Caffeine, nicotine, opium, and morphine are all examples of psychoactive drugs which, like cocaine, are formulated from plant alkaloids (Department of Health & Human Services [DHHS], 1984). From a public health perspective, cocaine is currently the drug of greatest national concern. While chewing coca leaves has been a practice for more than 2,000 years, the pure drug has only been available in the past century.

Cocaine is found in the leaves of two species of the coca shrub. The *erythroxylum coca* is the variety preferred by illegal manufacturers and requires a moist, tropical climate. *E. coca* is preferred because it yields the highest concentration of pure cocaine in its leaves which can be can be as high as 1.8% (Van Dyke & Byck, 1982). The *E. novogranatense* is cultivated in the drier, mountainous regions of Columbia. While it is not as potent a variety, the export value of the cocaine obtained from *E. novogranatense* is estimated to equal about half the value of the annual Columbian coffee crop (Van Dyke & Byck, 1982).

A variant of *E. novogranatense*, the *truxillense* or "Trujillo" variety, is grown in Peru. "Its leaves are harvested for legal export to the Stepan Chemical Co. in Maywood, NJ, where the cocaine is extracted for controlled pharmaceutical purposes and the remaining leaf material is prepared as a flavoring for Coca-Cola" (Van Dyke & Byck, 1982, p. 130).

History

When the Spanish *conquistadores* discovered the Inca empire, they found that use of the coca leaf was controlled by the Emperor himself. Coca leaf was dispensed only at important religious ceremonies or for special service to the Emperor. It was believed that Manco Capac, the royal son of the sun god, had sent it as a "gift from the gods to satisfy the hungry, fortify the weary, and make the unfortunate forget their sorrows" (Jones, 1953, p. 82). Among the highest honors the Inca could bestow was "the right to chew the coca leaf, which was prized far above the richest presents of silver or gold" (Blejer-Prieto, 1965, p. 701).

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Coca later came to serve more than a ceremonial function in ancient native American populations. Coca enabled early native Americans to endure the rigors of high altitude living as well as other unsuitable environments. Kleber and Gawin (1986) also noted that the "coca leaf contains a variety of nutrients, particularly vitamin B^1, riboflavin, and vitamin C, as well as protein. Approximately two ounces of coca leaves--the average amount used by the Incas daily--contained almost a minimum daily vitamin requirement" (p. 160). This was important because these early native Americans often had to exist in environments where "very hard work and limited food were characteristic" (Kleber & Gawin, 1986, p. 160).

The Spanish took over the Inca's coca leaves along with their empire. While the Spanish apparently did not use the drug themselves, they felt no reluctance to use coca leaf to control the native populations which were held

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in virtual slavery. It was noted that "under the effects of the coca leaf Indians worked harder, longer, and with less food. It also helped them, perhaps, to endure and forget, and even escape their misery" (Blejer-Prieto, 1965, p. 701). Initially, the Roman Catholic church opposed the use of coca leaf, however, the church "eventually initiated and maintained coca plantations itself (Kleber & Gawin, 1986, p. 160).

The use of coca leaves, unlike the use of tobacco and coffee beans, did not immediately become popular in Europe or North America. This is probably due to the failure of the coca leaf to retain its potency after drying and its unsuitability for cultivation in European climates. Since cocaine could not be grown and would not survive the months long trip by sail from South America to Europe, there was probably very little availability of the drug. According to Van Dyke and Byck (1982), cocaine would have to await the development of chemical processes for isolating the drug from the leaf before the it could be introduced in sufficiently large quantities into European cultures.

Cocaine was first used by Europeans in the nineteenth century when extract from the coca leaf was combined with various beverages. The most notable of these beverages was manufactured by a Corsican monk named Angelo Mariani and was known as "Mariani's wine" (Brecher, 1972). Mariani's wine was used to help "Christian ascetics withstand the pangs of hunger during prolonged fasts" (Brecher, 1972, p. 270). This mixture of cocaine and wine became so popular, that even Pope Leo XIII is reported to have used Mariani's wine (Brecher, 1972).

The German chemist Friedrich Gaedecke is credited with being the first to isolate cocaine from the coca leaf in 1855 (Van Dyke & Byck, 1982). It was the German chemist Albert Niemann, however, who was the first to describe the drug and to name it (Kleber & Gawin, 1986). This now meant that the active ingredient in the coca leaf was available in drug form and without the problems associated with storage.

Discovery of the pharmaceutical properties of cocaine soon followed its isolation. In 1859 Paolo Montegazzo declared coca leaves a "great new weapon against disease" (Eisworth, Smith, & Wesson, 1972). Montegazzo's endorsement of cocaine was so effusive, that he declared that he would "rather have a life span of 10 years with coca than one of one million centuries without it" (Petersen, 1977).

By the late 1800s, cocaine was undergoing considerable medical and pharmacological scrutiny. In 1884 enthusiastic reports about cocaine came to the attention of a young Viennese neurologist named Sigmund Freud. Freud found cocaine useful in treating his own depression and in treating pain and morphine addiction. Freud was so enthusiastic about the effects of cocaine, that he declared it a "magical drug" and began "collecting the literature for a song of praise to this magical substance" (Jones, 1953, p. 84).

Freud was a very vocal advocate of cocaine from about 1884 to 1887, when a friend he was treating for painful tumors became addicted to cocaine and developed a paranoid psychosis and hallucinations. Freud was also under considerable professional criticism from other medical authorities for his

advocacy of cocaine. In fact, hostility toward Freud became so great that Albrecht Erlenmeyer accused Freud of "having unleashed 'the third scourge of humanity' (after alcohol and the opiates)" (Van Dyke & Byck, 1982). Eventually, Freud discontinued all personal and medical use of cocaine. The irony of this episode is that were it not for cocaine's toxicity, Freud might not have directed his energies toward the development of psychoanalysis.

In 1885 John Styth Pemberton of Atlanta marketed a patent medicine called "French Wine Coca" which was reputed to be the ideal nerve tonic (Brecher, 1972). The following year, he added an extract of kola nutwhich is about 2% caffeineand renamed the product CocaCola. CocaCola was initially advertised as a "remarkable therapeutic agent" and as a "sovereign remedy" for a long list of ailments (Brecher, 1972).

By 1890 the addicting and psychosis producing nature of cocaine was wellunderstood and welldocumented. However, the drug still was largely unregulated and was a frequent ingredient in patent medicines used for the treatment of chronically swollen nasal passages (catarrh). "Cocaine has the effect of reducing mucous membrane swelling and thus enlarging the nasal and bronchial passages. This property no doubt first gave users the idea of *sniffing* [original emphasis] cocaine, a common form of cocaine use even today" (Brecher, 1972, p. 276).

The use of cocaine except as a local anesthetic was outlawed in 1914 with the Harrison Narcotic Act. From 1914 until the 1960s, the recreational use of cocaine was largely restricted to "jazz musicians, actors, and other members of the cultural avantgarde" (Kleber & Gawin, 1986, p. 161). In the late 1960s, cocaine enjoyed a renaissance of use for what would appear to be three primary reasons. First, because cocaine's withdrawal symptoms are relatively minor, it was mistakenly believed that cocaine was nonaddicting and, therefore, safe. Second, federal restrictions on the sale of amphetamines were making synthetic stimulants more difficult to obtain (Brecher, 1972). Finally, cocaine's extravagant price meant that to be able to afford cocaine was an indication of one's achievement and social status.

During the 1970s, cocaine use continued to escalate. By the late 1970s, the increase in cocaine use could only be described as explosive. According to a 1979 report, some 10 million Americans had taken cocaine within the preceding 12 months, compared with 10,000 people 20 years before (Van Dyke & Byck, 1982). The illegal importation of vast quantities of cocaine was rising dramatically from year to year in order to meet demand. In 1979 between 25,000 and 31,000 kilograms of cocaine entered the US illegally. This is in contrast with 1980, in which US cocaine imports were estimated to have been between 40,000 and 48,000 kilograms (Van Dyke & Byck, 1982).

In the early years of cocaine use, the price of cocaine was quite prohibitive. A kilogram of cocaine could cost as much as $20,000. By 1983, the price of a kilogram had plunged to $5,000 (Iyer, 1985), but has now risen to roughly $7,000 to $8,000 per kilogram. Currently, a gram of cocaine sells for between $50 and $100, while one to five rocks of crack cocaine sells for between $5 and $25. What is significant is that while the price of cocaine has

decreased over the last ten years, the purity of black market cocaine has more than doubled (Lieber, 1986).

Appearance

Cocaine comes as a white crystalline powder. However, a product called crack cocaine may come as an opaque crystal similar in size and shape to rock salt. The similarities are such that crack cocaine is sometimes referred to as rock cocaine. A third form of cocaine is known as coca paste. Coca paste is an intermediate step in the production of cocaine, and is becoming increasingly popular especially among poor people in the cocaine producing nations (Estroff, 1987; Siegel, 1987). Finally, because cocaine is soluble, it may also come as a clear liquid which is typically injected.

Administration and Absorption

Cocaine is highly water and fat soluble and, therefore, it is readily absorbed and distributed throughout the body (Jones, 1984). The effects of cocaine are determined by how rapidly it is absorbed into the bloodstream. The more rapidly the concentration rises, the more pleasurable the effect will be. However, peak concentrations which are high and rapid also produce the most dysphoria as blood concentrations decrease.

Route of administration seems to be more important in determining cocaine's absorption rather than dose. Certain routes of administration are more efficient than others, and can therefore produce higher peak concentrations more rapidly.

About 30% to 40% of the drug will be absorbed into the bloodstream over the course of an hour if the drug is taken *intranasally* (Jones, 1984). Initial uptake of the drug appears to be faster if the drug is taken intranasally (2 to 3 minutes) than if it is taken orally (8 to 10 minutes), and this may account for the more common practice of nasal administration among users (Jones, 1987). Peak concentrations for both oral and nasal cocaine are achieved in about 60 minutes (Jones, 1984).

Traditionally, injection of the highly water soluble cocaine hydrochloride had been the most efficient route of administration for delivering large concentrations of the drug rapidly into the bloodstream. The cocaine was either administered intravenously by itself or in combination with heroin (a *speedball*). Injection is difficult, however, and carries with it the added risk of infection. Recently, cocaine injection has given way to cocaine smoking as the preferred route for achieving rapid increases in blood concentrations.

Inhalation of cocaine smoke (*freebasing*) is a highly efficient mode of administration. Circulation from the lungs to the brain is approximately 6-7 seconds, while intravenously administered cocaine may take two to three times as long to reach the brain (Jones, 1987). Initially, it was thought that only freebasing could deliver such high concentrations, but it has recently been determined that smoking coca paste can deliver high concentrations of cocaine at very low cost (Siegel, 1989).

While the half-life of cocaine in the blood is from 45 to 90 minutes (Gawin & Ellinwood, 1988; Van Dyke & Byck, 1982), euphoric effects may last only a few minutes. There is often a "crash" or period of dysphoria and drug craving following cocaine administration (Kleber & Gawin, 1986). The intensity of this rebound is dependent on the rapidity with which peak plasma levels were achieved. The more rapidly peak concentrations were achieved, the shorter the effect and the deeper the crash. The severity of the crash also seems to be related to the intensity of use. Persons who have been using large amounts of cocaine over long periods of time (so called cocaine binges), appear to have more profound and longer lasting crashes (Extein & Dackis, 1987).

Psychological, Behavioral, and Pharmacological Effects

Cocaine is a powerful central nervous system and behavioral stimulant (Julien, 1988). This means that cocaine will stimulate both brain and behavioral activity in the user. The effects of cocaine on the brain, and especially the reward centers of the brain, are so powerful, that cocaine rivals heroin in its ability to foster dependence on the drug. It is believed that cocaine produces such a powerful and rapid dependency because it directly stimulates the pleasure centers of the brain responsible for the reinforcing properties of food, water, and sex (Extein & Dackis, 1987).

When applied topically, cocaine can act as a very potent local anesthetic. However, its use as an anesthetic has been supplanted by more effective drugs like *lidocaine* (Xylocaine) and *procain* (Novocaine). These drugs are considered to be superior to cocaine as local anesthetics for most purposes.

When cocaine is chewed, there are almost no psychological effects attendant to using the drug. The primary effects seem to be increased alertness and diminished appetite. When the drug is administered intranasally, intravenously, or smoked, a powerful psychological effect is produced consisting of *increased mental alertness, accompanied by feelings of enhanced competence and euphoria.* The effects of cocaine are not unlike those of another central nervous system stimulant, amphetamine. The chief difference between the two drugs would seem to be that the duration of action for amphetamine is much longer than that of cocaine (Julien, 1988).

Toxic Effects

While relatively small doses of cocaine can produce pleasurable effects, most acute toxic effects from cocaine appear to be "associated with the rapid onset of high plasma levels" (Jones, 1984, p. 42). Chronic administration of small doses has also been observed to produce responses normally expected only from a much larger dose. This is known as the *kindling effect*, whereby nerve pathways appear to become hypersensitive to the presence of the drug (Jones, 1984, 1987; Van Dyke & Byck, 1982). In humans, the toxic psychological response is characterized by increases in *irritability,*

restlessness, intense anxiety, hypervigilance, paranoid ideation, and suspicious behavior (Jones, 1984, 1987). Auditory, visual, or tactile hallucinations may also be experienced (Estroff, 1987), but are reported to be quite rare (Julien, 1988).

Overdose on cocaine is evidenced by *tremors, convulsions, elevated blood pressure, and death from cardiac or respiratory arrest* (Estroff, 1987). The lethal dose level for cocaine is reported to be 1,200 mg after oral ingestion, however, death from overdose has been caused by doses as low as 30 mg when the drug has been taken intranasally (Eiswirth, Smith, & Wesson, 1980). Jones (1984) reports that various authorities have estimated the maximum safe intranasal dosage of cocaine to be in the range of 100 to 300 mg. Since most intranasal users are reported to consume about 500 mg per day, they are operating within a "narrow margin between the dose of cocaine that will produce euphoria and a dose that will kill" (Eiswirth, Smith, & Wesson, 1980).

Tolerance does appear to raise the lethal dose level. It is important to remember, however, that the kindling effect may also lower the lethal dose level by making the individual hypersensitive to the drug (Jones, 1984, 1987). Because of the wide variability in the lethal dose level, Wetli (1987) concluded that it is impossible to state "what constitutes a 'safe' dose, especially in the recreational user" (p. 37). Jones (1984) felt that the lethal dose level also varied with route of administration and that death can occur as the result of cocaine administration by "virtually any route if the dose is large enough" (p. 46). Jones also stated that "death following cocaine administration may occur so rapidly that treatment is not available" (p. 46).

Less severe evidence of cocaine toxicity includes damage to or destruction of the nasal mucous membranes, perforation of the cartilage separating the nasal passages, malnutrition as a result of the appetite suppressant properties of cocaine, and dental problems resulting from neglect. It should be pointed out that all routes of administration pose some risk to the user; it is just that some routes are more dangerous than others (Estroff, 1987).

Is Cocaine Addicting?

For a drug to be considered addicting, three characteristics must be satisfied. First, using the drug must produce tolerance. Tolerance takes the form of requiring more of the drug with repeated use to achieve the desired effect. Second, the drug must be capable of producing psychological dependence. Psychological dependence is manifested by an intense craving for the effects of the drug to the point where the user prefers the drugged state over that of his or her normal state of functioning and will continue to use the drug in spite of the consequences. Third, the drug must produce physical dependence. This is evidenced by the presence of a withdrawal syndrome when the drug is discontinued. "Withdrawal symptoms represent the clinical manifestation of compensatory brain mechanisms that have developed in response to chronic drug use and are unmasked upon abrupt cessation of drug use" (Extein & Dackis, 1987, p. 75).

There is considerable debate as to whether cocaine produces physiological dependence as evidenced by a physical withdrawal syndrome. There are those who argue that dependence on cocaine is purely psychological. It would seem, however, that there is now considerable evidence of a physical withdrawal syndrome among cocaine addicts. What is different about cocaine withdrawal is that it is not as "florid or distinct as opioid or alcohol withdrawal" (Extein & Dackis, 1988, p. 76). Withdrawal appears to consist of "*low energy, depression, irritability, sleep disturbance, and craving for cocaine*" (authors' emphasis; Extein & Dackis, 1988, p. 76).

Intranasal users do not seem to be as prone to addiction in the classic sense of the term as are those who smoke cocaine. However, they may develop a very rapid and profound psychological dependence on the drug which is just as intense as addiction to opiates or alcohol. Those who smoke or freebase cocaine are much more likely to achieve physical dependence and appear to do so in a relatively short period of time. It should be noted, however, that intranasal use of cocaine does not provide any protection from dependency on cocaine, as there are as many intranasal users of cocaine entering treatment as there are freebasers.

The depression and dysphoria following regular use is what appears to fuel the compulsion to readminister cocaine. This crash can be quite uncomfortable for the user, and the user quickly learns that additional doses of cocaine can be used as a remedy for this depression (Gawin & Ellinwood, 1988; Julien, 1988). This compulsion to use does not appear to be limited to chronic users of the drug either. Cocaine users tend to continue to use the drug as long as it is available and it is the high price of cocaine that is the single most important variable regulating availability (Wise, 1984). It is not unlike eating potato chips or peanuts, most people find it difficult to stop after just one.

Treatment

"Whenever a new drug is introduced into a society without a well developed and functioning belief and support system, problems may be expected to arise" (DHHS, 1984). For our society, cocaine is such a drug. Because of cocaine's expense, severe psychological, medical, social, and occupational disruption can occur in the absence of extreme abuse or dependence (Kleber & Gawin, 1984, 1986). Consequently, individuals presenting themselves for treatment of cocaine abuse may constitute a widely varied group.

Kleber and Gawin (1984) note that "any cocaine user who finds that he cannot stop or significantly cut back his use in spite of the presence of problems arising from the use" is most probably in need of treatment (p. 111). Using this definition, persons in need of treatment may range from individuals who are episodic users to persons who are using the drug chronically and in large doses. It also means that any user, regardless of the intensity of the use or the route of administration, is at risk of developing a problem with cocaine which may require treatment.

Evaluation of cocaine abuse treatment has been sparse and no consensus exists regarding optimal treatment strategies (Kleber & Gawin, 1984, 1986). Much of what is available is based on clinical observation and treatment experience with other drugs of abuse. There are also no data available indicating what proportion of those who want to cease cocaine use are able to do so without treatment (Kleber & Gawin, 1984). Because so little data exists, it is difficult to say what conditions must be present to assist users in abstaining from cocaine. As a consequence, "no generally accepted or successful treatment has emerged" (Kleber & Gawin, 1984, p. 115). There are a number of treatments, however, which are in their initial stages and which do show promise.

Psychotherapy

Kleber and Gawin (1984) claim that almost all psychotherapeutic approaches to cocaine abuse are organized around three dimensions:

1. To help abusers recognize the deleterious effects of cocaine use and accept the need to stop.

2. To help abusers manage their impulsive behavior in general,and their cocaine use in particular.

3. To help abusers understand the functions that cocaine has played in their lives and to help abusers serve these functions without drugs. (p. 116)

Currently, psychotherapeutic approaches to cocaine abuse treatment are being employed primarily through self-help groups and structured treatment programs.

Self-help groups. Self-help groups offer group support and a 24 hour network of assistance. Typically, these self-help groups adopt a religious or spiritual tenor, are based on the 12 step model of Alcoholics Anonymous (AA), and espouse total abstinence from all psychoactive substances. The two self-help groups which frequently serve to meet the needs of cocaine abusers are Cocaine Anonymous (CA) and Narcotics Anonymous (NA). Self-help groups are powerful adjuncts to formal treatment and attendance at these meetings should be an integral part of any psychotherapeutic treatment approach.

Structured treatment programs. The goal of most structured treatment programs is to intervene in the individual's pattern of use by disrupting or suspending use and by blocking access to the user's supply. Such programs typically rely on the "liberal use of hospitalization" to achieve this goal (Kleber & Gawin, 1986, p. 175). Structured treatment programs emphasize indoctrination of the abuser in the harmful consequences which have been produced as a result of cocaine use and require abstinence from cocaine, alcohol, and all other psychoactive drugs as the only viable treatment outcome. While these programs are only able to claim moderate long-term success rates, Washton (1987) and Kleber and Gawin (1986) have identified a number of situations in which one would recommend hospitalization.

1. The patient is physically dependent on alcohol or other drugs.

2. Chronic free-base or intravenous use, as this type of use is most frequently out of control.

3. There is a concurrent severe medical or psychiatric condition complicating treatment.

4. The patient lacks motivation or family or social support.

5. Outpatient treatment has been attempted with repeated failures.

6. The patient has ready access to large amounts of cocaine.

Hospitalization has become quite controversial in the treatment of cocaine abuse. Many feel that hospitalization is far too expensive, that the recovery rates for inpatient treatment are not appreciably better than those of outpatient programs, that it is disruptive of families and careers, and that inpatient treatment does not teach the abuser how to abstain from cocaine upon re-entry into the "real world." Consequently, many treatment programs are being developed to provide comprehensive services strictly on an outpatient basis.

Pharmacotherapy

Kleber and Gawin (1984) report that there is sufficient evidence to indicate that "enduring neurophysiologic changes" or abnormalities occur following chronic cocaine use (p. 118). It is reasonable to assume, then, that certain pharmacotherapies may exist which might help to correct cocaine induced neural abnormalities and support abstinence in those abusers seeking treatment. Research into the effectiveness of various pharmacotherapies is currently underway, and the most promising are reviewed below.

Tricyclic Antidepressants or TCAs. Cocaine addiction appears to produce psychological and behavior depression as well as low energy levels which are similar to the symptoms found in psychiatrically depressed patients. It has been shown that the same antidepressant medication which is effective in cases of psychiatric depression can help reverse the depression and lack of energy found in abstinent cocaine users. Some of the advantages to antidepressant therapy include: (a) low incidence of adverse effects, (b) low toxicity, (c) high patient acceptance, and (d) little or no abuse potential (Kleber & Gawin, 1987).

Lithium carbonate. Cocaine produces neural and behavioral stimulation which resembles that of a manic episode. Kleber and Gawin (1987) report that there are a number of case studies in which lithium carbonate has been found to block the stimulation produced by acute cocaine intoxication. They report that it has also been found that cocaine use decreased during the course of lithium treatment. While lithium therapy would seem to hold some promise, the authors indicated that there are several problems associated with lithium. First, lithium is a toxic mineral and blood levels must be monitored to protect against overdose. Second, lithium's ability to block the effects of cocaine may

be overridden by large doses. Finally, lithium may only be effective in those cases where patients use cocaine to selfmedicate a preexisting bipolar or cyclothymic disorder.

Methylphenidate or MPH: MPH, or *ritalin*, produces central nervous system effects similar to those of cocaine. It has been suggested that maintenance on MPH could raise the dose level of cocaine necessary to produce euphoria. Since the euphoria producing effect of cocaine would be blunted, there would be no incentive to continue to use cocaine. MPH maintenance for cocaine users would, therefore, be similar in theory to methadone maintenance for heroin addicts. While the theory for this approach is credible, MPH presents several problems for this type of treatment. First, MPH carries some abuse potential among this patient population. Second, Kleber and Gawin (1987) report that MPH "actually appeared to increase both cocaine craving and cocaine use by inducing a mild euphoria that acted as a conditioned cue for the more intense onset and degree of euphoria produced by cocaine" (p. 123). Finally, it may only be effective for those patients who use cocaine as a form of selfmedication for a residual attention deficit disorder. While drug substitution or maintenance therapies certainly hold promise for the future of cocaine treatment, it would appear that this approach will have to await a safer, more effective drug.

Guidelines for cocaine abuse treatment

While there are many different forms and modalities of cocaine abuse treatment, there also seem to be a number of considerations which are common to all treatments. Anyone developing a treatment plan or program for a cocaine abuser should keep these considerations in mind. First, the program should encourage users to change their "playmates, playground, and playthings." This means that users should avoid frequenting those places where they used in the past, they should break off contact with former dealers and friends who are still using, and they should get rid of any drug paraphernalia or other artifacts which have become associated with cocaine use. The important point here is to "make the drug 'psychologically' unavailable since it is so difficult to make it physically unavailable" (Kleber & Gawin, 1986, p. 177).

Second, because cocaine can only be detected in the blood for about two days after last use, urinalysis should be conducted frequently. Urinalysis should be conducted to deter future cocaine abuse and for early detection of lapses (Kleber & Gawin, 1986). Washton (1987) states that "the purpose of urine testing is *not* to catch the patient in a lie. Rather, urine testing is an extremely useful treatment tool that helps to counteract denial, promotes self-control over drug impulses, and provides an objective indicator of treatment progress" (original emphasis; p. 109).

Third, the program should require that cocaine abusers abstain from all mood altering drugs and not just cocaine. Kleber and Gawin (1986) and Washton (1987) have identified four reasons for requiring total abstinence among cocaine abusers.

1. Complete abstinence provides abusers with "the widest margin of safety from potential relapse" (Washton, 1987, p. 108).

2. Drugs which have been used in conjunction with cocaine may become conditioned cues which can trigger a desire for cocaine.

3. Users may become dependent on other drugs.

4. An abuser's ability to resist offers of cocaine may be significantly reduced while under the influence of other drugs.

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